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CASE REPORT

Infection of an Internal Carotid Artery Plaque with Vancomycin Resistant Enterococcus

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Introduction

The first reported case of infection of a carotid plaque with vancomycin resistant enterococcus (VRE) is described.

Case Report

A 68 year old male, with a history of left hemicolectomy for vesico-colic fistula (diverticular disease (1990)), coronary bypass (1992), partial gastrectomy for neoplasia (1996), non-insulin dependent diabetes and hypertension, presented with multiple left carotid territory TIAs. Duplex revealed a >80% ipsilateral stenosis. At endarterectomy, a 1 cm "yellow-green" saccular lesion projected from the lateral aspect of the internal carotid artery (ICA), 2 cm above the bifurcation (Fig. 1). Because infection was considered possible, the bifurcation was resected en-bloc before blood flow was restored via a Pruitt-Inahara shunt (Ideas for Medicine Ltd). A reversed saphenous vein bypass was performed without complication.

There was a severe stenosis at the origin of the ICA. Within the plaque, there was a pool of liquefied debris (Fig. 2). This was in continuity with the "aneurysm", but not the lumen of the ICA. The wall of the saccular projection retained all three arterial layers with thinning of elastin tissue. Microbiological culture of the plaque debris yielded VRE (*Enterococcus faecium*), sensitive to Linezolid. The patient was given a six-week course of oral linezolid (600 mg bd, Pharmacia Ltd) with regular haematological and biochemical surveillance. Serial clinical and duplex ultrasound review has shown no evidence of re-infection at 18 months. Post-operative echocardiography showed no evidence of endocarditis.

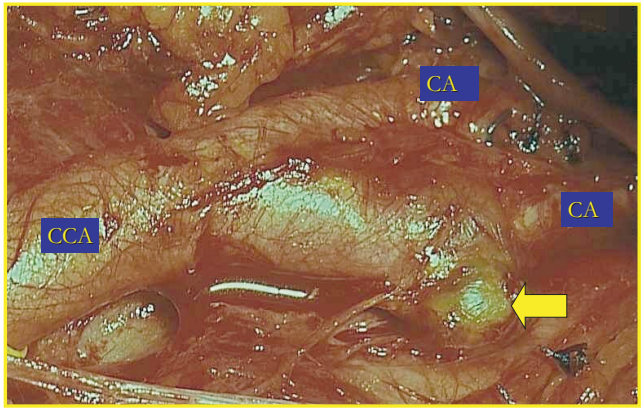

Fig. 1. Operative photograph of "mycotic" aneurysm (yellow arrow) on the internal carotid artery (ICA). (CCA = common carotid artery, ECA = external carotid artery).

Fig. 2. Schematic diagram of location of necrotic debris culturing VRE in relation to luminal surface and aneurysm.

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Discussion

The above case either represents a "mycotic" aneurysm or infection of a carotid plaque. ICA "mycotic" aneurysms are extremely rare and usually follow haematogenous *Staphylococcus aureus* infection. Associations with *Escherischia coli*, *Salmonella* and *Pseudomonas* species have been described, but never VRE.[1]

VRE was isolated in 1986.[2] It accounts for 20% of all non-ICU, nosocomial enterococcal isolates in the U.S.A.[3] and poses a major management problem in immunocompromised patients, especially transplant patients. Despite its increasing prevalence and resistance to virtually all antibiotics, VRE colonisation usually poses few problems to healthy individuals.[3]

The aneurysm/infected plaque was not identified pre-operatively, probably because the sac was encased in an inflammatory peri-arterial/jugular vein reaction within the neck. Primary sources for the infection include endocarditis,[4] pharyngeal infection or the lower gastrointestinal tract, which is the commonest site for colonisation with VRE.[3] On review, there was nothing in the patient's history to suggest that a cardiac or pharyngeal source was likely.

One possible explanation is occult infection of the atherosclerotic plaque during an episode of bacteraemia with secondary aneurysm formation. The patient had undergone a hemicolectomy for vesico-colic fistula secondary to diverticular disease in 1990. Inevitably, diverticulae in the residual colon will progress and may have been the portal for a subsequent VRE bacteraemia. There is no evidence, however, that this infection was responsible for the onset of his TIAs. Macroscopically and histologically, the zone of infection was separate from the luminal surface and there was no surface disruption to the plaque.

The post-operative management of this patient was facilitated by the sensitivity of the VRE to Linezolid. Unfortunately, reports of Linezolid-resistant VRE are already emerging.[5]

References

1 Jebara VA, Acar C, Dervanian P *et al*. Mycotic aneurysms of the carotid arteries: Case report and review of the literature. *J Vasc Surg* 1991; **14**: 215–219.
2 Uttley AHC, Collins CH, Naidoo J, George RC. Vancomycin resistant enterococci. *Lancet* 1988; **I**: 57–58.
3 Cates JA, Gelabert HA. Primary arterial infections. In: Moore WS, ed. *Vascular Surgery: A Comprehensive Review* (fourth edition). W.B. Saunders, Philadelphia, 1993; pp 147–159.
4 Murray BE. Vancomycin resistant enterococcal infections. *N Engl J Med* 2000; **342**: 710–720.
5 Herrero IA, Issa NC, Patel R. Nosocomial spread of Linezolid-resistant, vancomycin-resistant *Enterococcus faecium*. *N Engl J Med* 2002; **346**: 867–869.

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